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Fig. 1. The matching result of 2-D gel map of proteins from PBMC of HCV infected patients and healthy persons.

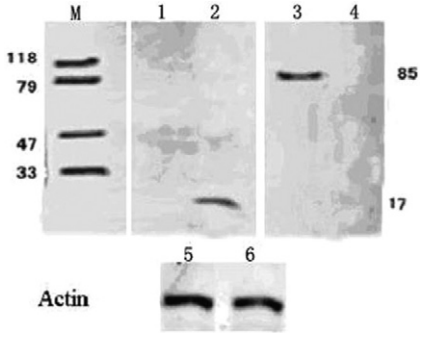

Fig. 2. Detecting F protein and PI3K by Western blot analysis. M: Protein marker; 1, 4, 5: pcDNA3.0-HepG2; 2, 3, 6: pcDNA3.0-F-HepG2.

significant influence on cell signal pathway. HCV-F protein can activate PI3K protein and its signal cascade pathway. Therefore, HCV-F protein may plays an important role in HCV pathogenesis.

OL-003 Secretion of hepatitis C virus E2 protein could enhance neutralization antibodies induced by DNA vaccine

Zhi-hui Chen[1], Yi-min Tong[2], Zhong-tian Qi*[,2]. [1]*Department of Infectious Diseases, Changhai Hospital, Second Military Medical University, Shanghai, China;* [2]*Department of Microbiology, Second Military Medical University, Shanghai, China*

Objective: To explore the feasibility of induction of neutralization antibodies against HCV infection by DNA vaccines.
Methods: Two expression plasmids of HCV envelope 2 protein were constructed, plasmid pCI-1b746 encoding full length of E2 protein, and plasmid pCI-1b661 encoding hydrophobic carboxyl terminal truncated E2. 293T cells were transfected with either of both plasmids, respectively, and intracellular and secreted E2 protein were analyzed by Western blot or immunofluorescence. Then the plasmids were used to immunize BALB/c mouse intramusclarly. The sera antibodies against E2 and hypervariable region 1 (HVR1) were detected by ELISA and the neutralization activity of the sera were assayed with HCV pseudotype particle (HCVpp).
Results: Both plasmids could express HCV E2 protein, the expression product of pCI-1b746 could not secrete, while that of pCI-1b661 could secrete into culture medium. The secretion of HCV E2 protein enhanced antibody response elicited by DNA vaccines in mice significantly. Sera from pCI-1b661 immunized mice showed stronger neutralization activity than that from pCI-1b746 immunized mice. For sera from pCI-1b661 immunized mice, the neutralization activity against HCVpp were positive correlation with anti-HVR1 antibodies levels.
Conclusions: Secretion of E2 protein by DNA vaccine can enhance the production of neutralization antibodies against HCVpp infection and the neutralizing activity is depended on the presence of HVR1 antibodies.

OL-004 Suppression of liver fibrosis is associated with the decrease of transforming growth factor-β1 and increase of matrix metalloproteinase-1 expression after interferon-α therapy in chronic hepatitis C patients

Yun-ru Li*. *Beijing Ditan Hospital, Beijing, China*

Aim: To evaluated relationship of the changes of transforming growth factor-β1 (TGF-β), matrix metalloproteinase-1 (MMP-1) and tissue inhibitors of matrix metalloproteinase-1 (TIMP-1) expression with liver fibrosis in liver tissue after IFN-α therapy in patients with chronic hepatitis C.
Methods: Eleven patients with chronic hepatitis C treated by IFN-α were divided into two groups on the outcome of therapy, including a complete responder group (CR) and a non-responder group (NR). Liver biopsy specimens were stained with hematoxylin and eosin (HE) and Masson's trichrome staining. Liver fibrosis was semiquantitated by Modified Ishak scoring system.
Results: There was a significant reduction in Ishak fibrosis score in SVR group ($P<0.05$). The TGF-β1 expression was reduced and the MMP-1 expression was elevated in SVR groups after IFN-α therapy ($P<0.05$). The ratio of the density of MMP-1 to TIMP-1 expression increased in SVR group after IFN-α therapy compared with NR group ($P<0.05$). The reduction of fibrosis score correlated significantly with that of TGF-β1 expression in all patients ($R=0.744$, $P<0.01$). The increase of MMP-1 expression after IFN-α therapy significantly correlated with the decrease of fibrosis ($R=-0.925$, $P<0.05$) and HAI ($R=-0.837$, $P<0.05$) scores in SVR group.
Conclusion: IFN-α improves liver fibrosis in hepatitis C patients by decreasing TGF-β1 expression, as well as by increasing MMP-1 expression to degrade fibrosis.

OL-005 Immunohistochemical study of hepatic oval cells in fetal liver and chronic C hepatitis

Maria Comanescu*[,1], Violeta Comanescu[2]. [1]*University Clinical Hospital of Bucharest, Romania;* [2]*County Clinical Hospital of Craiova, Romania*

Background: Hepatic oval cells differentiate into two types of cells, hepatocytes and biliary cells. The aim of this study was to identify oval cells in fetal liver and in adult liver from patients with chronic C hepatitis.
Method: This study was performed using two study group. The first included 10 human fetal livers from embryos from therapeutical abortions with gestational ages ranging betwwen 10 and 21 weeks. The second group included 30 liver biopsies from patients with viral C hepatitis. The liver specimens were fixed in formaldehyde, embedded in paraffin and cut at 5 μm. Slides were stained using standard (HE, VG) and immunohistochemical stainings (CK19, CD45, alfaSMA and desmin).
Results: All cases were examined by light microscopy. Oval cells were identified as small cells with basophilic cytoplasm and small oval nuclei, located in periportal areas, and in association with fibrosis and inflammation. They were positive for CK19 and negative for CD45. Desmin was positive in fetal hepatocytes and negative in adult hepatocytes. Alfa SMA was positive in periportal cells and negative in hepatocytes and biliar ducts. In chronic C hepatitis alfa SMA was positive in isolated cells in the areas of necrosis.